Nutritional Aspects of Amenorrhea in the Female Athlete Triad

Joan E. Benson, Kathryn A. Engelbert-Fenton, and Patricia A. Eisenman

Female athletes experience a high incidence of menstrual abnormalities. This has critical health consequences because amenorrheic athletes are at greater risk of developing osteopenia and bone injury compared to normally menstruating athletes or nonathletic normally cycling females. Female performers and athletes are also at risk for developing disordered eating behaviors. There appears to be a connection between menstrual dysfunction, athletic training, and disordered eating, but how they relate is not fully understood. In this paper we explore how low calorie intakes, nutritional inadequacies, vegetarianism, low body fat stores, and specific training behaviors may contribute to the abnormal menstrual patterns seen in this population. Recommendations for the detection and prevention of eating and training problems and consequent menstrual abnormalities are included.

Key Words: disordered eating, vegetarianism, weight loss, menstrual dysfunction

With the passage of Title IX legislation in 1972, women have enjoyed the benefits of increased participation in sports activities. In most high schools and colleges, women’s athletic programs are growing and thriving. But along with the benefits, there has been increasing concern about the medical problems that are being observed among this active female population. The young athlete, driven to excel in her chosen sport, may be at risk of developing a triad of interrelated problems: disordered eating, amenorrhea, and osteoporosis (49, 50), termed the female athlete triad. While concerns about eating disorders among female athletes have been long-standing, it is only in recent years that the connection between eating disorders and amenorrhea and resulting loss of bone mass has come into public view.

Although studies have focused on the medical consequences of anorexia nervosa and bulimia in the athletic population, there is a spectrum of disordered eating behaviors, ranging from severe (clinically diagnosed anorexia nervosa or bulimia) to mild (poor nutritional habits) into which many athletes fall. Although their disordered eating patterns may not fit the criteria for clinical diagnosis, many

J.E. Benson is with the Department of Oncological Sciences, K.A. Engelbert-Fenton is with the Division of Foods and Nutrition, and P.A. Eisenman is with the Department of Exercise and Sports Sciences, University of Utah, Salt Lake City, UT 84112.
female athletes are still at risk for developing serious medical problems and progressing into an eating disorder. The prevalence of disordered eating in female athletes has been reported to be high—32 to 62% (35). Female athletes also experience a high incidence of menstrual abnormalities, such as secondary amenorrhea, irregular cycles, and anovulatory cycles (30). Between 25% and 40% of highly trained endurance athletes report fewer than three menses per year (9, 30, 40, 56). A connection appears to exist between abnormal eating behaviors and amenorrhea; in fact, amenorrhea is among the diagnostic criteria for anorexia nervosa. However, it may also occur in lesser degrees of disordered eating (49). This connection has critical health consequences, because amenorrheic athletes and performers are at greater risk of developing osteopenia compared to normally menstruating athletes or nonathletic normally cycling females (13, 22). This decrease in bone mass increases the likelihood of debilitating lower extremity stress fractures (40, 45) and more devastating fractures of the hip and spine.

Disordered eating may be expressed as a collection of behaviors, including low calorie intakes, weight loss, body fat loss, excessive exercise, vegetarianism, or fat restriction (49). Attempts to explain athletic amenorrhea in the past have focused on these behaviors as potential etiological factors. However, the evidence linking these factors to menstrual dysfunction has been contradictory and confusing. To add to the confusion, there has been a lack of uniformity regarding the definition of amenorrhea.

In this paper we review how weight loss, body fat loss, exercise, nutritional inadequacy, and vegetarianism may be linked to menstrual abnormalities, and we discuss the health consequences of abnormal menses in athletes. Finally, we present recommendations for detection, treatment, and prevention of this triad of disorders.

**Causes of Athletic Amenorrhea**

**Amenorrhea**

Amenorrhea means the “absence of menstrual bleeding.” Primary amenorrhea refers to absence of menstruation in women who have never had a menstrual period (menarche after 16 years of age). Secondary amenorrhea refers to the absence of menstrual cycles in women who have had established periods. There is no universal agreement about the number of months of no cycle required to diagnose amenorrhea, but it is usually between 3 and 6 consecutive months (3). Oligomenorrhea is a menstrual cycle length between 35 and 90 days, and “irregularity” is defined as either amenorrhea or oligomenorrhea (63). It is thought that any degree of menstrual dysfunction may have medical consequences (3).

**Weight Loss**

In several early studies, simple weight loss was suggested to be a factor in producing amenorrhea among sedentary women (26). Twenty years ago, Frisch and McArthur (29) proposed a “critical weight concept,” stating that there is a set amount of body weight required to initiate and maintain menses. In their study, and in subsequent research (9, 16, 30), dancers and athletes experiencing irregular menses had significantly lower body weights for height than their counterparts reporting regular cycles. The nature of the relationship between weight loss and menstrual dysfunction...
is not well understood. Women with weight loss–associated amenorrhea have been reported to have abnormalities in gonadotropin and estrogen secretion (38) and peripheral steroid metabolism (24). Other possible mechanisms for amenorrhea suggested by Frisch and McArthur (29) depend on the role of adipose tissue in the aromatization of estrogens and the potential effects of estrogens on hypothalamic regulatory centers. Fishman et al. (24) theorized that as body weight decreases, estrogen metabolism shifts from the normal 16-hydroxylation to 2-hydroxylation (catechol estrogens). Catechol estrogens are steroids that structurally resemble estrogens but have only a weak estrogenic effect (46).

**Low Body Fat Levels**

Body fatness has also been proposed as a predictor of menstrual dysfunction. Frisch and McArthur (29) hypothesized that body fat must reach 17% of body weight before menstruation begins and must reach a minimum of 22% for menstruation to resume when secondary amenorrhea results from weight loss. However, other authors (14, 21, 37, 56) have found regularly menstruating subjects with percent body fat levels well below the critical level proposed by Frisch and McArthur (29) and have noted no body composition differences between regularly menstruating athletes and those with amenorrhea. However, it should be noted that there was variation in the measurement methods employed by these researchers. Kaiserauer et al. (37) compared amenorrheic runners, regularly menstruating runners, and regularly menstruating sedentary controls for plasma hormone levels, physical characteristics, and nutrient intakes. While there were no differences in body fatness, body weight, or degree of exercise between the two groups, the amenorrheic subjects consumed less fat, red meat, and total calories than did the regularly menstruating subjects. Deuster et al. (21) found a similar lack of difference in body fat measures of amenorrheic and eumenorrheic runners. However, the dietary fat intake of amenorrheic runners was significantly lower than that of eumenorrheic runners (66 vs. 97 g/day), and fiber intake was higher in the amenorrheic group.

So while it appears that menstrual disturbance is not a function of total body fat as a percentage of body weight, these data do suggest that a combination of low body weight, variable nutrient or calorie intake, and exercise exerts synergistic effects in the development of menstrual problems. In addition, there is the possibility that it is not total body fatness, but rather degree of fatness at specific body sites, that directly influences gonadal function.

**Loss of Specific Fat Stores**

In their review of the weight regulation practices of athletes, Brownell et al. (10) suggested that specific fat deposits, rather than total body fat, may be key determinants of menstrual function. Research has shown that metabolic activity of the adipose tissue of women differs by region during pregnancy and lactation (55). Lipolytic activity of adipose tissue is higher in the femoral region than in the abdominal region in pregnant and lactating women. In addition, the femoral region exhibits higher lipoprotein lipase (LPL) activity in both nonpregnant and pregnant women but is particularly pronounced during pregnancy.

These findings suggest that femoral adipose deposits (fat found in the hips, buttocks, and thighs) play a key role in the female reproductive process, such that
when these fat stores fall below a certain level, pregnancy and lactation may not be viable. Depletion of these specific fat deposits in female athletes could possibly account for menstrual abnormalities, by providing a signal that the body cannot sustain fertility. A recent study has suggested that patterns of fat distribution differ between athletes with and without menstrual irregularities (33). In this study, eumenorrheic college gymnasts had significantly greater fat stores in the lateral thigh than their amenorrheic counterparts, although total percent body fat measures did not differ. This could explain the conflicting results of studies exploring the relationship between body fat and amenorrhea.

**Excessive Exercise**

Exercise, alone or in conjunction with decreased body weight, may contribute to the development of menstrual abnormalities. Several studies (16, 19, 28, 68) have noted menstrual irregularities especially during the performance or competitive season when exercise loads increase. Exercising subjects who also experienced weight loss had significantly more delayed menses than subjects who maintained their weight during test periods (11). It is likely that increased exercise affects menses only when it is accompanied by weight loss. A hypothesized mechanism by which exercise may affect the menstrual cycle involves the hypothalamic–pituitary–adrenal axis (43). Research on athletes has shown that cortisol levels in amenorrheic athletes are elevated about 25% while sedentary and cyclic athletes experience little change in a 24-hr period (43). Loucks described past research on animals showing that the gonadotropin-releasing hormone pulse generator in the hypothalamus can be inhibited by corticotropin-releasing hormone, the hormone that stimulates the release of adrenocorticotropic hormone (ACTH), which then activates the adrenal cortex during stress, such as stress of exercise. Other animal research has found gonadotropin secretion by ACTH to be inhibited only in the presence of intact adrenals, suggesting a direct effect of cortisol on the reproductive system (31).

**Nutritional Inadequacy**

Insufficient caloric intake (7, 22), even in the absence of physical training, has been proposed as a factor contributing to menstrual dysfunction (23, 58). The relationship between nutritional intake and exercise training remains elusive. Broocks et al. (7) monitored cyclic ovarian function with daily hormonal measurements and serial ultrasound determinations of follicular development in a group of 17 recreational athletes and 13 sedentary controls. While 11 of the 13 controls demonstrated estradiol (E2) and progesterone (P4) concentrations as well as normal luteal phase lengths (9 days or more), only 10 of 17 athletes satisfied these criteria. The eumenorrheic and amenorrheic athletes had similar aerobic capacities and similar training levels (63 min/day for amenorrheic vs. 53 min/day for normally menstruating athletes). There was no difference in caloric consumption between the groups, as calculated from food diaries. Since the athletes were expending more energy due to training, the reported caloric intake values indicated that as a group they had failed to increase energy intake appropriately. Thus, the researchers speculated that inadequate nutritional adaptation may have been a factor contributing to menstrual dysfunction.
This lack of difference in caloric intake has also been noted by others comparing female athletes and sedentary controls (18, 45) as well as those comparing eumenorrheic and amenorrheic athletes (22, 51). An apparently negative energy balance without a change in body weight could be explained by a significant reduction in metabolic rate during nontraining hours or by increased metabolic efficiency during exercise. However, it is possible that small sample sizes contributed to the failure to find statistically significant differences in calorie intakes between groups. Additionally, the number of intake days averaged to determine caloric intake was not consistent among these studies, and these studies did not report how subjects were instructed to record dietary data.

The reduction in resting metabolic rate (RMR) that accompanies caloric restriction is well documented (20, 34, 47, 66). In most cases, the decline in RMR can be attributed solely to losses of lean body mass. Although exercise is commonly believed to offset the reduction in RMR that results from calorie restriction, this is not always the case (34, 53, 66). Evidence that some amenorrheic athletes consume less energy than regularly menstruating athletes, coupled with the discovery that exercise does not necessarily lead to an elevation in RMR, suggests that an athlete with menstrual irregularities may be characterized by negative energy balance and reduced RMR. This hypothesis was supported by a recent study which documented that the RMR of amenorrheic runners was significantly lower than that of eumenorrheic runners and sedentary controls (44) although calorie intakes were the same. Loucks and Callister found suppressed thyroid function in a group of exercising females who restricted their calorie intakes (44). This discovery raises interesting questions about metabolic changes occurring in amenorrheic women.

Apparently not all women athletes experience the same metabolic changes. Some studies report no differences in training load or energy intake for amenorrheic and eumenorrheic athletes (18, 51). These findings contradict the premise that caloric deficit is the only factor that causes menstrual dysfunction. Caloric deficits could work in a synergistic fashion with training intensity and impact ovarian function, or it might be that alterations in the composition of macronutrient consumption are the determining variable. In a study with 6 normal-weight women (42), a reduction in dietary fat from 40% of 1,800 total calories to 25% of calories resulted in a significant shift away from the 16-hydroxylated estrogen metabolites toward the catechol estrogens. Anderson et al. (5), also noted that alterations in the composition of macronutrients result in shifts in 16-hydroxylase and 2-hydroxylase activity. These differences in metabolic pathways are of potential significance because of the peripheral activity of the metabolites that result from 16- and 2-hydroxylation. C-2 products have virtually no uterotropic activity, while C-16 products are potent uterotropic agents (25).

There are data showing that athletes do consume diets lower in fat content than many sedentary control groups, which would then result in an increase in C-2 products and might explain menstrual function differences between athletes and sedentary subjects. However, there are reports of groups of athletes whose dietary fat intakes do not differ yet whose menstrual function responses to increased training loads do differ, contradicting the premise that percentage of dietary fat is the only regulating factor (61). This, along with many of the other seemingly contradictory data, suggests that individual variability with respect to metabolism interacts with external variables such as diet and training to induce menstrual dysfunction.
Vegetarianism

Another of the possible external variables influencing menstrual function is vegetarianism. Brooks et al. (8) noted that more female runners with amenorrhea were vegetarians ($p < .05$) compared to those who regularly menstruated. Kaiserauer et al. (37) reported that a greater percentage of amenorrheic runners (25%) than eumenorrheic runners (11%) were considered to be vegetarians. Slavin, Lutter, and Cushman (60) reported high rates of amenorrhea in vegetarian athletes and speculated that components of these diets, such as trace elements or plant hormones, may affect menstruation.

Lloyd et al. (41) examined the effects of several nutritional factors on menstrual function and bone density in collegiate athletes and concluded that increased dietary fiber intake is associated with menstrual dysfunction and a lower bone density in this cohort. In a study of nonathletes, Snow et al. (62) found that oligomenorrheic women consumed significantly more dietary fiber and significantly less saturated fat than their eumenorrheic counterparts. How fiber affects menstrual function is unclear. Studies have shown that vegetarian women who consumed twice as much fiber as nonvegetarian women had reduced circulating estrone and estradiol levels and significantly greater fecal excretion of estrogens (32). Dietary fiber increases fecal bulk, which in turn decreases the concentration of intestinal B-glucuronidase, an enzyme that hydrolyzes the bile acid–steroid complex, an event necessary for the reabsorption of estrogens (1). Some fibers may also bind nonpolar estrogens in the intestinal lumen, reducing their reabsorption (59). Preliminary results in the large “Finlandia” study have revealed positive correlations between intake of total fiber and plasma concentration of sex hormone binding globulin (SHBG) and negative correlations between fiber and plasma percentage of free estradiol (%FE) (1). The result could be a reduction in the bioavailability of the hormone for reproductive and other functions. Plant lignins and isoflavones have weak estrogenic activity and may compete with estradiol for binding sites. It has also been suggested that lignins and isoflavones reduce the biological activity of estradiol (2). A recent study suggested that isoflavone intake from soy was a factor in causing amenorrhea in a group of women (15).

The inability of research to consistently show that calorie deficits, body fat losses, or excessive exercise cause menstrual dysfunction may be explained by the failure to consider adjustments to metabolic rate and/or specific body fat stores that may accompany exercise training in certain individuals. In addition, while the evidence linking dietary fat and calorie intake levels to menstrual dysfunction is inconsistent, the failure to consider fiber and vegetable intake in these analyses may explain the inconsistency of findings. In all likelihood, athletic amenorrhea results from a combination of factors.

Health Consequences

While the possible mechanisms of athletic amenorrhea are of interest, the real importance of menstrual abnormalities in athletic females is related to the effects of low levels of certain hormones (irregular cycles being an indicator) on bone mineralization and density and on injury risk. Both anorexic and amenorrheic women suffer from premature bone loss that may be rapid and has been shown to be partially irreversible. Decreased bone density results in a higher risk of stress
fractures and other more serious fractures. Amenorrhea has been related to scoliosis and even increased risk of soft tissue injury. Whether or not amenorrhea in young athletes is a risk factor for cardiovascular disease, as it is in postmenopausal women, is a subject of recent research.

**Identification, Prevention, and Treatment of Triad Disorders**

How do we identify athletes whose behaviors place them at risk for bone loss, and more importantly, how do we intervene once they are identified? Athletes from all sports can develop disordered eating, amenorrhea, and resulting complications. But it appears that those most likely to be affected are athletes from three distinct groups of sports: “appearance sports” such as gymnastics, figure skating, diving, and ballet; sports in which low body weight is considered advantageous such as distance running, sports climbing, and cycling; and “weight category” sports such as judo and crew (54). Anorexia nervosa and bulimia nervosa represent the extreme forms of disordered eating patterns (4) and are often the ones that concern coaches and physicians the most. It is becoming increasingly apparent, however, that there is a spectrum of abnormal eating varying from mild to severe (6, 65). These patterns of disordered eating, such as the relentless effort to eliminate all fat from the diet or restricting oneself to only one meal a day, can have a negative impact on health and performance despite their lack of classification as a disease state. Health care professionals and athletic support personnel should not restrict themselves to the clinical definitions of eating disorders when evaluating individuals for problems of body weight, eating behaviors, or excessive training. The term “disordered eating” has been applied to the female athlete triad to include any pattern of abnormal intake that could result in nutritional or psychological problems (71). Since low fat intakes, weight loss or calorie deficits (including those caused by excess exercise), and vegetarianism have been related to menstrual problems, athletes who exhibit these behaviors should be monitored for menstrual dysfunction. In addition, athletes, coaches, parents, athletic administrators, training staff, sport science personnel, and team physicians need to be provided a program of education about the female athlete triad (50). Such a program should to the following:

1. Give athletes the message that sports participation is congruous with the physical and mental well-being of women.
2. Encourage a change in thinking on the part of coaches and athletic administrators to “win at all costs.”
3. Teach young athletes that healthy eating habits are consistent with good performance and that poor habits will in the long run hurt one’s ability to perform well.
4. Educate athletes, parents, and coaches on the elements of a healthy eating plan, emphasizing adequate calories, protein, fat, calcium, and iron.
5. De-emphasize weigh-ins and body composition measurements as a basis for making judgments about an athlete’s performance. Rather, emphasize the importance of feeling strong, having energy and stamina, feeling good about oneself, and eating a healthy diet.
6. Dispel myths and misinformation about weight, dieting, and performance. There is little evidence that thinness improves performance. In fact, caloric
restriction can cause fatigue, anemia, and electrolyte imbalances and can ultimately worsen performance.

7. Educate athletes, parents, and coaches on the effects of puberty: establishing normal menses, developing secondary sex characteristics, and increasing body fat. Help athletes recognize these changes as normal and desirable.

8. Encourage athletes to monitor and report changes in menstrual cycles to a parent, coach, or team physician. Establish a contact person (e.g., athletic trainer) with whom the athletes can discuss concerns and who can refer the athlete for medical evaluation if appropriate.

In addition, coaches and parents should watch for the warning signs of serious eating disorders and take immediate action if an athlete is suspected of having a serious eating problem. The American College of Sports Medicine has published guidelines to assist in the management of such athletes (3).

**Medical Screening**

Female athletes can be screened for problems related to disordered eating, amenorrhea, and decreased bone mass during the preparticipation physical (36, 50). Complete weight and menstrual histories along with open-ended questions about dieting and weight control behavior should be included. If the athlete is age 14 and prepubertal, or age 16 and premenarchal, she should be evaluated for primary amenorrhea. If she is menarchal but has been without menses for 3–6 months, she should be screened for secondary amenorrhea. Since caloric restriction or other nutritional deficiencies can contribute to either primary or secondary amenorrhea, a work-up for these conditions should include an evaluation for disordered eating. The athlete with a history of stress fractures should be evaluated for menstrual dysfunction and nutrition status. After athletes have passed the initial screening procedures, continued monitoring throughout the year is recommended.

**Treatment**

Because bone loss can occur quickly in young women who stop menstruating, treatment should be sought as soon as problems are suspected. However, the appropriate treatment remains controversial. In treating amenorrhea, the goals are twofold: to prevent bone loss and to reestablish a normal menstrual cycle (52). The length of time required to induce normal menses through lifestyle measures may be longer than a physician is comfortable waiting, especially if the athlete has been amenorrheic for some time. Then, the issue of elevating the circulating levels of estrogen with exogenous hormone therapy must be considered (52, 58).

If the amenorrheic athlete is willing and able to make lifestyle changes, it is recommended that she decrease her training activity by 10–20%, increase her calorie intake, gain 2–3% of her body weight, add resistance training to workouts, and supplement with 1,500 mg/day of calcium (52). In addition, fiber supplements should be avoided. Measures of bone density may be obtained if the athlete has been amenorrheic for 1 year or longer, or if she cannot be convinced to make lifestyle changes without evidence of bone loss. Follow-up bone scans can then be performed yearly to monitor changes (64).
Estrogen replacement therapy should be considered if the athlete has been unwilling to make lifestyle changes or if those changes have not been effective in reestablishing menses; if the athlete has been amenorrheic for longer than 6 months (or longer than 2 years if she is an adolescent); if estradiol levels are very low; or if there is a history of stress fractures (52). Estrogen replacement therapy in young athletes is controversial. Current recommendations are based on data from postmenopausal women and may not be appropriate for this population. Little is known about the long-term risks associated with estrogen replacement in young athletes (50, 52). But since bone loss progresses rapidly and irreversibly, this treatment measure should be strongly considered. Regardless of the treatment decision, regular follow-up and evaluation of eating patterns, training schedule, and menstrual function should continue as long as the athlete participates in sport.

References


